10/861,428

EAST Search History

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	4921	544/253.ccls. and TGFB or (transforming adj growth adj factor-beta)	US-PGPUB; USPAT; USOCR	OR	OFF	2007/01/12 14:06
L2	0	544/253.ccls. and transforming adj growth adj factor-beta	US-PGPUB; USPAT; USOCR	OR	OFF	2007/01/12 14:06
L3	0	544/253.ccls. and (transforming adj growth adj factor-beta)	US-PGPUB; USPAT; USOCR	OR	OFF	2007/01/12 14:06
L4	0	544/253.ccls. and (TGFB)	US-PGPUB; USPAT; USOCR	OR	OFF	2007/01/12 14:07
L5	0	544/253.ccls. and TGFB	US-PGPUB; USPAT; USOCR	OR	OFF	2007/01/12 14:07
L6	467	(544/253).CCLS.	US-PGPUB; USPAT; USOCR	OR	OFF	2007/01/12 14:09
L7	0	(544/253andcompound).CCLS.	US-PGPUB; USPAT; USOCR	OR	OFF	2007/01/12 14:09
L8	229	l6 and (transforming growth factor)	US-PGPUB; USPAT; USOCR	OR	OFF	2007/01/12 14:10
L9	2	l6 and (transforming adj growth adj factor)	US-PGPUB; USPAT; USOCR	OR	OFF	2007/01/12 14:22
L10	154	(514/258.1).CCLS.	US-PGPUB; USPAT; USOCR	OR	OFF	2007/01/12 14:22
L11	0	l10 and (transforming adj growth adj factor)	US-PGPUB; USPAT; USOCR	OR	OFF	2007/01/12 14:25
L12	0	L10 and (transforming adj growth adj factor)	US-PGPUB; USPAT; USOCR	OR	OFF	2007/01/12 14:26
L13	5	L10 and (TGF)	US-PGPUB; USPAT; USOCR	OR	OFF	2007/01/12 14:26
L14	0	L10 and (TGF.clm.)	US-PGPUB; USPAT; USOCR	OR	OFF	2007/01/12 14:26

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NEWS	2		"Ask CAS" for self-help around the clock
NEWS	3	OCT 23	The Derwent World Patents Index suite of databases on STN has been enhanced and reloaded
NEWS	4	OCT 30	CHEMLIST enhanced with new search and display field
NEWS	5	NOV 03	JAPIO enhanced with IPC 8 features and functionality
NEWS	6	NOV 10	CA/CAplus F-Term thesaurus enhanced
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NEWS	8	NOV 20	CAS Registry Number crossover limit increased to 300,000 in additional databases
NEWS	9	NOV 20	CA/CAplus to MARPAT accession number crossover limit increased to 50,000
NEWS	10	DEC 01	CAS REGISTRY updated with new ambiguity codes
NEWS	11	DEC 11	CAS REGISTRY chemical nomenclature enhanced
NEWS	12	DEC 14	WPIDS/WPINDEX/WPIX manual codes updated
NEWS	13	DEC 14	GBFULL and FRFULL enhanced with IPC 8 features and functionality
NEWS	14	DEC 18	CA/CAplus pre-1967 chemical substance index entries enhanced with preparation role
NEWS	15	DEC 18	CA/CAplus patent kind codes updated
NEWS	16	DEC 18	MARPAT to CA/CAplus accession number crossover limit increased to 50,000
NEWS	17	DEC 18	MEDLINE updated in preparation for 2007 reload
NEWS	18	DEC 27	CA/CAplus enhanced with more pre-1907 records
NEWS	19	JAN 08	CHEMLIST enhanced with New Zealand Inventory of Chemicals

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NEWS X25	X.25 communication option no longer available

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                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.21       0.21

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FILE LAST UPDATED: 11 Jan 2007  (20070111/ED)

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=> s transforming growth factor beta
         67297 TRANSFORMING
       1316611 GROWTH
          4444 GROWTHS
       1318879 GROWTH
                  (GROWTH OR GROWTHS)
       1011346 FACTOR
        911973 FACTORS
       1594774 FACTOR
                  (FACTOR OR FACTORS)
       1428937 BETA
          1328 BETAS
       1429013 BETA
                  (BETA OR BETAS)
L1       31500 TRANSFORMING GROWTH FACTOR BETA
                  (TRANSFORMING(W)GROWTH(W)FACTOR(W)BETA)

=> s l1 and inhibit?
       1892670 INHIBIT?
L2       14096 L1 AND INHIBIT?

=> s l2 and disease?
       1036739 DISEASE?
L3        4543 L2 AND DISEASE?

=> s l3 and inflammator?


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        170803 INFLAMMATOR?
L4        1028 L3 AND INFLAMMATOR?

=> s l4 and arthritis?
         44491 ARTHRITIS?
L5         148 L4 AND ARTHRITIS?

=> s l5 and py<2003
      22868738 PY<2003
L6           59 L5 AND PY<2003

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'DISORDER/' IS NOT A VALID FIELD CODE
For a list of field codes for the current file, enter "HELP SFIELDS"
at an arrow prompt (=>).

=> s l6 and disorder?
        442986 DISORDER?
L7           21 L6 AND DISORDER?

=> d ibib abs hitstr tot

L7   ANSWER 1 OF 21  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:         2002:793447  CAPLUS
DOCUMENT NUMBER:          137:304813
TITLE:                    Modulators of hedgehog signaling pathway for treatment
                          of T-cell-mediated diseases
INVENTOR(S):              Lamb, Jonathan Robert; Hoyne, Gerard Francis; Dallman,
                          Margaret Jane; Champion, Brian Robert
PATENT ASSIGNEE(S):       Lorantis Limited, UK
SOURCE:                   PCT Int. Appl., 154 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.           KIND   DATE        APPLICATION NO.         DATE
     ---------------      ----   --------    --------------------    --------
     WO 2002080952         A2    20021017    WO 2002-GB1666          20020409 <--
     WO 2002080952         A3    20040108
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
             GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
             LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,
             PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ,
             UA, UG, US, UZ, VN, YU, ZA, ZM, ZW
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
             KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB,
             GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA,
             GN, GQ, GW, ML, MR, NE, SN, TD, TG
     AU 2002247847         A1    20021021    AU 2002-247847          20020409 <--
     EP 1401469            A2    20040331    EP 2002-716928          20020409
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
     JP 2004534743         T     20041118    JP 2002-578991          20020409
     US 2004126359         A1    20040701    US 2003-682230          20031009
PRIORITY APPLN. INFO.:                       GB 2001-8872         A  20010409
                                             GB 2001-8873         A  20010409
                                             WO 2002-GB1666       W  20020409
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AB Use of a modulator of a Hedgehog signaling pathway, or a modulator of a pathway which is a target of the Hedgehog signaling pathway in the preparation of a medicament for treatment of a disease or disorder associated with a T-cell mediated disease or disorder.

L7 ANSWER 2 OF 21 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:777707 CAPLUS
DOCUMENT NUMBER: 137:273242
TITLE: Carbon monoxide-generating compounds for treatment of vascular, inflammatory and immune disorders
INVENTOR(S): Buelow, Roland; Woo, Jacky
PATENT ASSIGNEE(S): Sangstat Medical Corporation, USA
SOURCE: PCT Int. Appl., 59 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002078684	A2	20021010	WO 2002-US10115	20020401 <--
WO 2002078684	A3	20021219		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2442457	A1	20021010	CA 2002-2442457	20020401 <--
EP 1381354	A2	20040121	EP 2002-757910	20020401
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
CN 1507348	A	20040623	CN 2002-809432	20020401
JP 2004526739	T	20040902	JP 2002-576950	20020401
PRIORITY APPLN. INFO.:			US 2001-280526P P	20010330
			WO 2002-US10115 W	20020401

AB Methods and compns. are provided for treating vascular disease and modulating the inflammatory and immune processes using carbon monoxide-generating compds., including methylene chloride. The compds. are capable of inhibiting the proliferation of vascular smooth muscle cells, protecting the vasculature against oxidative stress and injury, modulating the activity of various immune system cells, inhibiting the production of pro-inflammatory cytokines and enhancing production of anti-inflammatory cytokines, thereby being effective in the treatment of conditions associated with adverse proliferative or inflammatory responses. Methods for extending the survival of an organ transplant and inhibiting chronic rejection in a recipient are also provided.

L7 ANSWER 3 OF 21 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:713810 CAPLUS
DOCUMENT NUMBER: 138:265336
TITLE: Metallothionein suppresses collagen-induced arthritis via induction of TGF-β and down-regulation of proinflammatory mediators

Erich Leeser

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AUTHOR(S): Youn, J.; Hwang, S.-H.; Ryoo, Z.-Y.; Lynes, M. A.; Paik, D.-J.; Chung, H.-S.; Kim, H.-Y.
CORPORATE SOURCE: Institute of Biomedical Science and Department of Anatomy and Cell Biology, College of Medicine, Hanyang University, Seoul, S. Korea
SOURCE: Clinical and Experimental Immunology (2002), 129(2), 232-239
CODEN: CEXIAL; ISSN: 0009-9104
PUBLISHER: Blackwell Science Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Metallothionein is a low mol. weight, cysteine-rich, stress response protein that can act as an antioxidant and as an immunosuppressive agent in instances of antigen-dependent adaptive immunity. In this context, the authors assessed the therapeutic potential and mechanisms of action of metallothionein in a collagen-induced arthritis model. Repeated administration of metallothionein-I + II during the course of disease dramatically reduced the incidence and severity of the disease. Joint tissues isolated from boostered paws of metallothionein-I + II-treated mice expressed significantly reduced levels of proinflammatory mediators, such as tumor necrosis factor (TNF)-α and cyclooxygenase-2, when compared with those of control-treated mice. Lymph node cells obtained from metallothionein-I + II -injected mice exhibited a significant decrease in the proliferative response and a remarkable increase in tumor growth factor (TGF)-β production in response to type II collagen. Taken together, these results suggest that metallothionein-I + II promote the development of type II collagen-specific, TGF-β-producing cells to antagonize the expansion of arthritogenic cells. This could lead to local suppression of inflammatory responses by inhibiting the expression of proinflammatory mols. Thus, this study demonstrates the suppressive effects of metallothionein on collagen-induced arthritis, and indicates that there may be a potential therapeutic application for manipulation of metallothionein during the treatment of autoimmune disorders.

REFERENCE COUNT: 54 THERE ARE 54 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 4 OF 21 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:594822 CAPLUS
DOCUMENT NUMBER: 137:154857
TITLE: Preparation of nicotinamide biaryl derivatives as inhibitors of PDE4 isozymes
INVENTOR(S): Chambers, Robert James; Magee, Thomas Victor; Marfat, Anthony
PATENT ASSIGNEE(S): Pfizer Products Inc., USA
SOURCE: PCT Int. Appl., 224 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002060875	A1	20020808	WO 2001-IB2341	20011206 <--
WO 2002060875	A8	20030731		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,

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LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,
PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA,
UG, US, UZ, VN, YU, ZA, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB,
GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA,
GN, GQ, GW, ML, MR, NE, SN, TD, TG

CA 2436535	A1	20020808	CA 2001-2436535		20011206 <--
EP 1355884	A1	20031029	EP 2001-273556		20011206
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR					
EE 200300360	A	20031215	EE 2003-360		20011206
BR 2001016852	A	20040225	BR 2001-16852		20011206
HU 200400637	A2	20040628	HU 2004-637		20011206
JP 2004520386	T	20040708	JP 2002-561026		20011206
CN 1518542	A	20040804	CN 2001-823071		20011206
NZ 526453	A	20050128	NZ 2001-526453		20011206
US 2002193612	A1	20021219	US 2002-62813		20020131 <--
US 6649633	B2	20031118			
ZA 2003004894	A	20040624	ZA 2003-4894		20030624
US 2004048903	A1	20040311	US 2003-613988		20030702
US 6953810	B2	20051011			
BG 108038	A	20040730	BG 2003-108038		20030728
NO 2003003397	A	20030919	NO 2003-3397		20030730
PRIORITY APPLN. INFO.:			US 2001-265492P	P	20010131
			WO 2001-IB2341	W	20011206
			US 2002-62813	A3	20020131

OTHER SOURCE(S): MARPAT 137:154857
GI

* STRUCTURE DIAGRAM TOO LARGE FOR DISPLAY - AVAILABLE VIA OFFLINE PRINT *

AB The title compds. [I; g = 0-1; j = 0-1; provided that when j = 0, n must be 2; k = 0-1; m = 0-2; n = 1-2; W1 = 0, SOt (t = 0-2), NR3; W2 = OCR9R10, or absent; Y = CR1, NOk (k = 0-1); R9, R10 = H, F, CF3, etc.; or R9 and R10 are taken together, but only in the case where m = 1, to form a spiro moiety; R7, R8 have the same meaning as R9, R10 except that one of them must be H; R1, R2 = H, F, Cl, etc.; R3 = H, alkyl, Ph, etc.; R4-R6 = H, F, Cl, etc.; Q1 = Ph, benzodioxyl, etc.; Q2 = biaryl moiety], useful as inhibitors of PDE4 in the treatment of diseases regulated by the activation and degranulation of eosinophils, especially asthma, chronic bronchitis, and chronic obstructive pulmonary disease, were prepared E.g., a multi-step synthesis of the amide II, starting from Me 3-bromobenzoate and 4-formylbenzeneboronic acid, was given. Compds. I showed anti-inflammatory activity at 0.0001 μM to 20.0 μM in whole blood assay for LTE4.
REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 5 OF 21 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:314727 CAPLUS
DOCUMENT NUMBER: 136:339498
TITLE: Methods for treating IL-18 mediated disorders
INVENTOR(S): Sims, John E.; Mohler, Kendall M.; Born, Teresa L.
PATENT ASSIGNEE(S): Immunex Corporation, USA
SOURCE: PCT Int. Appl., 53 pp.

Erich Leeser

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CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002032374	A2	20020425	WO 2001-US32460	20011017 <--
WO 2002032374	A3	20020919		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2002098185	A1	20020725	US 2002-981421	20020118 <--

PRIORITY APPLN. INFO.: US 2000-241408P P 20001018

AB The invention pertains to methods for treating medical disorders characterized by elevated levels or abnormal expression of IL-18 by administering an IL-18 antagonist, such as soluble IL-18 receptor, a soluble IL-18 binding protein and/or an antibody.

L7 ANSWER 6 OF 21 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:290814 CAPLUS
DOCUMENT NUMBER: 136:308541
TITLE: Interleukin-1 Hy2 polypeptides, polynucleotides and antibodies for prognosis, diagnosis and treatment of interleukin 1-mediated diseases
INVENTOR(S): Ballinger, Dennis G.; Pace, Ann M.; Lin, Hai Shan
PATENT ASSIGNEE(S): Hyseq, Inc., USA
SOURCE: U.S., 52 pp., Cont.-in-part of U.S. 6,175,532.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6372892	B1	20020416	US 2000-522964	20000310 <--
WO 2000071719	A1	20001130	WO 2000-US14144	20000522 <--

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RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

EP 1179068	A1	20020213	EP 2000-937687	20000522 <--

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO

US 6365726	B1	20020402	US 2000-578458	20000522 <--

PRIORITY APPLN. INFO.: US 1999-316086 A2 19990520
US 1999-316081 A 19990520

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	US 2000-522964	A	20000310
	WO 2000-US14144	W	20000522

AB The present invention provides novel nucleic acids encoding IL-1 Hy2, a novel member of the interleukin 1 receptor antagonist family, novel polypeptides encoded by these nucleic acids, antibodies, antisense DNA and RNA, oligonucleotide primers and probes, and others. These IL-1 Hy2 polypeptides, polynucleotides, and antibodies are useful for diagnosis, prognosis and therapy of IL-1-mediated diseases, e.g. sepsis and associated conditions, endotoxic shock, cytokine-induced shock, thrombosis, acute pancreatitis, rheumatoid or reactive arthritis, chronic inflammatory arthritis, vasculitis, lupus, immune complex glomerulonephritis, pancreatic cell damage from diabetes mellitus type 1, allograft and xenograft transplantation, graft vs. host disease, inflammatory bowel disease, inflammation associated with pulmonary disease, other autoimmune disease or inflammatory disease, acute or chronic myelogenous leukemia, ovarian carcinoma, etc. IL-1 Hy2 may be used concurrently with agents that inhibit the production or activity of interleukin 1 (e.g. GM-CSF, IL-4, IL-10, IL-13 and TGF-β) or other antiinflammatory agents (e.g. IL-1Ra, UL-1Ra-like protein IL-1 Hy1, anti-TNF, corticosteroids, immunosuppressive agent).

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 7 OF 21 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2002:251954 CAPLUS
DOCUMENT NUMBER: 136:278157
TITLE: Polynucleotides encoding IL-1 Hy2 polypeptides
INVENTOR(S): Ballinger, Dennis G.; Ford, John; Ho, Alice Suk-Yue; Lin, Hai Shan; Pace, Ann M.
PATENT ASSIGNEE(S): Hyseq, Inc., USA
SOURCE: U.S., 62 pp., Cont.-in-part of U.S. Ser. No. 522,964. CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
US 6365726	B1	20020402	US 2000-578458	20000522	<--
US 6339141	B1	20020115	US 1999-316081	19990520	<--
US 6372892	B1	20020416	US 2000-522964	20000310	<--
PRIORITY APPLN. INFO.:			US 1999-316081	A2 19990520	
			US 2000-522964	A2 20000310	
			US 1999-316086	A2 19990520	

AB The present invention provides novel nucleic acids encoding IL-1 Hy2, a novel member of the Interleukin-1 Receptor Antagonist family, the novel polypeptides encoded by these nucleic acids and uses of these and related products. These polypeptides and polynucleotides, as well as antibodies and antisense DNA or RNA are useful for diagnosis, prognosis, and therapy of disorders mediated by IL-1, IL-18 and/or IL-12, e.g. sepsis and associated conditions, thrombosis, acute pancreatitis, rheumatoid or reactive arthritis, chronic inflammatory arthritis, vasculitis, lupus, etc. Methods for identification of compds. that modulate the expression of activity of the polynucleotides and/or polypeptides of the invention are also provided.

REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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L7 ANSWER 8 OF 21 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:241369 CAPLUS
DOCUMENT NUMBER: 136:261819
TITLE: Coupling of peripheral tolerance to endogenous IL-10 promotes effective modulation of T cells and ameliorates autoimmune disease
INVENTOR(S): Zaghouani, Habib
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 84 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE	
US 2002038002	A1	20020328	US 2001-873901		20010604	<--
CA 2416656	A1	20020404	CA 2001-2416656		20010604	<--
AU 2001065417	A5	20020408	AU 2001-65417		20010604	<--
EP 1292621	A1	20030319	EP 2001-939953		20010604	
EP 1292621	B1	20060920				
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR						
JP 2004509978	T	20040402	JP 2002-531215		20010604	
AT 340193	T	20061015	AT 2001-939953		20010604	
PRIORITY APPLN. INFO.:			US 2000-209527P	P	20000605	
			EP 2001-939953	A	20010604	
			WO 2001-US40834	W	20010604	

AB Immunomodulating agents comprising at least one Fc receptor ligand and at least one immunosuppressive factor are provided as are methods for their manufacture and use. The immunomodulating agents may be in the form of polypeptides or chimeric antibodies and preferably incorporate an immunosuppressive factor comprising a T cell receptor agonist or antagonist. The compds. and compns. of the invention may be used to selectively suppress the immune system to treat symptoms associated with immune disorders such as allergies, transplanted tissue rejection and autoimmune disorders including autoimmune diabetes, rheumatoid arthritis and multiple sclerosis.

L7 ANSWER 9 OF 21 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2001:575814 CAPLUS
DOCUMENT NUMBER: 136:165344
TITLE: Degradative pathways in tissues of the temporomandibular joint: Use of in vitro and in vivo models to characterize matrix metalloproteinase and cytokine activity
AUTHOR(S): Puzas, J. Edward; Landeau, Jean-Marie; Tallents, Ross; Albright, Jeffries; Schwarz, Edward M.; Landesberg, Regina
CORPORATE SOURCE: Departments of Orthopaedics, University of Rochester School of Medicine and Dentistry, Rochester, NY, 10032, USA
SOURCE: Cells Tissues Organs (2001), 169(3), 248-256
CODEN: CTORFB; ISSN: 1422-6405
PUBLISHER: S. Karger AG
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Identification of a small animal model that undergoes pathol. temporomandibular joint (TMJ) degeneration would represent a significant

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research tool. To date however, no such model has been described. The authors therefore have investigated the pathol. and immunohistochem. features of the TMJ of a transgenic mouse that overexpresses the human form of TNFα. The TMJ of this animal appears to undergo changes that resemble arthriditics of temporomandibular dysfunction. Furthermore, the disk and articular cells express MMP9 and IL-1. Future work should validate this animal model as one that would have utility for the study of TMJ disorders. Maintenance of connective tissues in joints such as the TMJ is a normal process that allows for the reconstitution of important anat. features. This maintenance involves both the removal and resynthesis of structural proteins such as collagens, elastins and proteoglycans. An imbalance in the pathways for degradation and synthesis can lead to the degeneration of joint tissues. The authors describe the presence of a matrix metalloproteinase, MMP9 (92 kDa gelatinase), in TMJ disk and articular cells that likely function in the degradative process. Addnl., the authors show that this enzyme is under the control of pro-inflammatory cytokines whereby TGFβ and IL-1 stimulate and PGE2 inhibits its activity.

REFERENCE COUNT: 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 10 OF 21 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2001:466579 CAPLUS
DOCUMENT NUMBER: 136:165454
TITLE: Anticytokine gene therapy of autoimmune diseases
AUTHOR(S): Prud'homme, Gerald J.; Lawson, Brian R.; Theofilopoulos, Argyrios N.
CORPORATE SOURCE: The Department of Pathology, McGill University, Montreal, QC, H3A2B4, Can.
SOURCE: Expert Opinion on Biological Therapy (2001), 1(3), 359-373
CODEN: EOBTA2; ISSN: 1471-2598
PUBLISHER: Ashley Publications Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review. Viral and nonviral gene therapy vectors have been successfully employed to deliver inflammatory cytokine inhibitors (anticytokines), or anti-inflammatory cytokines, such as transforming growth factor β -1 (TGF-β1), which protect against exptl. autoimmune diseases. These vectors carry the relevant genes into a variety of tissues, for either localized or systemic release of the encoded protein. Administration of cDNA encoding soluble IFN-γ receptor (IFN-γR)/IgG-Fc fusion proteins, soluble TNF-α receptors, or IL-1 receptor antagonist (IL-1ra), protects against either lupus, various forms of arthritis, autoimmune diabetes, or other autoimmune diseases. These inhibitors, unlike many cytokines, have little or no toxic potential. Similarly, TGF-β1 gene therapy protects against numerous forms of autoimmunity, though its administration entails more risk than anticytokine therapy. We have relied on the injection of naked plasmid DNA into skeletal muscle, with or without enhancement of gene transfer by in vivo electroporation. Expression plasmids offer interesting advantages over viral vectors, since they are simple to produce, non-immunogenic and nonpathogenic. They can be repeatedly administered and after each treatment the encoded proteins are produced for relatively long periods, ranging from weeks to months. Moreover, soluble receptors which block cytokine action, encoded by gene therapy vectors, can be constructed from non-immunogenic self elements that are unlikely to be neutralized by the host immune response (unlike

monoclonal antibodies [mAbs]), allowing long-term gene therapy of chronic inflammatory disorders.
REFERENCE COUNT: 99 THERE ARE 99 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 11 OF 21 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2001:319747 CAPLUS
DOCUMENT NUMBER: 134:331637
TITLE: Use of GDNF for treating corneal defects
INVENTOR(S): Hanke, Michael; Kruse, Friedrich; Paulista, Michael; Pohl, Jens
PATENT ASSIGNEE(S): Biopharm Gesellschaft Zur Biotechnologischen Entwicklung Von Pharmaka m.b.H., Germany
SOURCE: PCT Int. Appl., 60 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001030375	A2	20010503	WO 2000-EP10674	20001030 <--
WO 2001030375	A3	20020321		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2385929	A1	20010503	CA 2000-2385929	20001030 <--
EP 1223966	A2	20020724	EP 2000-983097	20001030 <--
EP 1223966	B1	20030502		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL				
JP 2003512433	T	20030402	JP 2001-532792	20001030
AT 238806	T	20030515	AT 2000-983097	20001030
PT 1223966	T	20030930	PT 2000-983097	20001030
ES 2198367	T3	20040201	ES 2000-983097	20001030
US 2003166537	A1	20030904	US 2002-132069	20020424

PRIORITY APPLN. INFO.:

EP 1999-121597	A	19991029
WO 2000-EP10647	A1	20001030
WO 2000-EP10674	W	20001030

AB The present invention relates to the use of a glial cell line-derived growth factor (GDNF) or a functionally active derivative or part thereof and/or an agonist which substitutes the functional activity of GDNF, and/or a nucleic acid containing at least a nucleotide sequence encoding the primary amino acid sequence of GDNF or the functionally active derivative or part thereof and/or of the agonist for the manufacture of a pharmaceutical composition for epidermal and stromal wound healing.

L7 ANSWER 12 OF 21 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2001:265459 CAPLUS
DOCUMENT NUMBER: 134:290751
TITLE: Recombinant single-chain receptor antagonist proteins and their use in treatment of inflammatory disorders

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INVENTOR(S): Halkier, Torben; Schambye, Hans Thalsgard; Okkels, Jens Sigurd; Andersen, Kim Vilbour; Nissen, Torben Lauesgaard; Soni, Bobby; Jeppesen, Claus Bekker; Van Den Hazel, Bart
PATENT ASSIGNEE(S): Maxygen Aps, Den.
SOURCE: PCT Int. Appl., 123 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001025277	A1	20010412	WO 2000-DK563	20001006 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1226173	A1	20020731	EP 2000-965860	20001006 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL				
US 2004014948	A1	20040122	US 2003-444691	20030523

PRIORITY APPLN. INFO.:

DK 1999-1438	A	19991007
DK 1999-1855	A	19991223
DK 2000-1119	A	20000720
US 1999-160820P	P	19991021
US 2000-174655P	P	20000106
US 2000-225723P	P	20000816
US 2000-684720	B1	20001006
WO 2000-DK563	W	20001006

AB The invention relates to a single-chain oligomeric protein antagonist which binds to an extracellular ligand-binding domain of a cellular receptor of a type requiring binding of an oligomeric ligand to two or more receptor subunits to be activated, the protein comprising at least two, typically structurally homologous, receptor-binding sites of which at least one is capable of binding to a ligand-binding domain of the cellular receptor and at least one is incapable of effectively binding to a ligand-binding domain of the cellular receptor, whereby the single-chain oligomeric protein is capable of binding to the receptor, but incapable of activating the receptor; as well as to nucleotide sequences encoding such single-chain oligomeric proteins, expression vectors comprising such a nucleotide sequence, recombinant host cells comprising such a nucleotide sequence or expression vector, methods for producing the nucleotide sequences and proteins, pharmaceutical compns. comprising the single-chain oligomeric protein, and use of the single-chain oligomeric protein for the production of medicaments and in therapy. A preferred single-chain antagonist according to the invention is a TNF-α antagonist. Thus, a single-chain TNF-α protein comprising of 3 human TNF-α chains connected by linker peptides was produced with Saccharomyces cerevisiae and shown to be an agonist of the TNF-α receptor. The same TNF-α trimer containing Y87R mutations in the first and third copies of TNF-α was also prepared This was shown to be a partial TNF-α agonist and a competitive antagonist of the TNF-α receptor.

REFERENCE COUNT: 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS

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RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 13 OF 21 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2000:688272 CAPLUS
DOCUMENT NUMBER: 133:280563
TITLE: Human antibodies that bind human IL-12 and methods for producing
INVENTOR(S): Salfeld, Jochen G.; Roguska, Michael; Paskind, Michael; Banerjee, Subhashis; Tracey, Daniel E.; White, Michael; Kaymakcalan, Zehra; Labkovsky, Boris; Sakorafas, Paul; Friedrich, Stuart; Myles, Angela; Veldman, Geertruida M.; Venturini, Amy; Warne, Nicholas W.; Widom, Angela; Elvin, John G.; Duncan, Alexander R.; Derbyshire, Elaine J.; Carmen, Sara; Smith, Stephen; Holtet, Thor Las; Du, Fou Sarah L.
PATENT ASSIGNEE(S): Basf A.-G., Germany; Genetics Institute Inc.; et al.
SOURCE: PCT Int. Appl., 377 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000056772	A1	20000928	WO 2000-US7946	20000324 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2365281	A1	20000928	CA 2000-2365281	20000324 <--
NZ 513945	A	20010928	NZ 2000-513945	20000324 <--
BR 2000009323	A	20020108	BR 2000-9323	20000324 <--
EP 1175446	A1	20020130	EP 2000-918396	20000324 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
HU 200200575	A2	20020629	HU 2002-575	20000324 <--
TR 200102715	T2	20020923	TR 2001-2715	20000324 <--
JP 2002542770	T	20021217	JP 2000-606632	20000324 <--
US 6914128	B1	20050705	US 2000-534717	20000324
NZ 529571	A	20060331	NZ 2000-529571	20000324
TR 200503572	T2	20060421	TR 2005-3572	20000324
ZA 2001007774	A	20021220	ZA 2001-7774	20010920 <--
NO 2001004605	A	20011126	NO 2001-4605	20010921 <--
BG 106027	A	20020628	BG 2001-106027	20011018 <--
US 2005004354	A1	20050106	US 2004-884830	20040701
AU 2005200515	A1	20050303	AU 2005-200515	20050207
AU 2006225302	A1	20061026	AU 2006-225302	20061006

PRIORITY APPLN. INFO.:		
US 1999-126603P	P	19990325
AU 2000-39216	A3	20000324
US 2000-534717	A3	20000324
WO 2000-US7946	W	20000324
AU 2005-200515	A3	20050207

AB Human antibodies, preferably recombinant human antibodies, that specifically bind to human interleukin-12 (hIL-12) are disclosed. Preferred antibodies have high affinity for hIL-12 and neutralize hIL-12

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activity in vitro and in vivo . An antibody of the invention can be a full-length antibody or an antigen-binding portion thereof. The antibodies, or antibody portions, of the invention are useful for detecting hIL-12 and for inhibiting hIL-12 activity, e.g., in a human subject suffering from a disorder in which hIL-12 activity is detrimental. Nucleic acids, vectors and host cells for expressing the recombinant human antibodies of the invention, and methods of synthesizing the recombinant human antibodies, are also encompassed by the invention.

REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 14 OF 21 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2000:144772 CAPLUS
DOCUMENT NUMBER: 132:189689
TITLE: Bioreductive conjugates for drug targeting
INVENTOR(S): Adams, Ged; Blake, David; Naughton, Declan; Stratford, Ian
PATENT ASSIGNEE(S): Theramark Limited, UK; Adams, Margaret
SOURCE: PCT Int. Appl., 48 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 2000010610	A2	20000302	WO 1999-GB2606		19990819 <--
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM					
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG					
AU 9954296	A1	20000314	AU 1999-54296		19990819 <--
PRIORITY APPLN. INFO.:			GB 1998-18027	A	19980819
			GB 1998-18156	A	19980820
			WO 1999-GB2606	W	19990819

OTHER SOURCE(S): MARPAT 132:189689

AB The use of a bioreductive conjugate comprised of a noncytotoxic bioreductive moiety having linked thereto at least one therapeutic agent, and salts thereof, is disclosed for the healing of wounds and the treatment of fibrotic disorders, ulcerative colitis, inflammatory bowel disease, epilepsy, cardiovascular reperfusion injury, cerebral reperfusion injury, hypertension, cystic fibrosis, psoriasis, para-psoriasis, peptic ulcers, gastric ulcers, duodenal ulcers, diabetic ulcers dementia, oncol., AIDS, rheumatoid arthritis, diabetes, and ischemia. Various specific conjugates for treating these conditions are also disclosed.

L7 ANSWER 15 OF 21 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1999:487222 CAPLUS
DOCUMENT NUMBER: 131:120861
TITLE: Artificial proteoglycans for drug targeting and other therapeutic applications
INVENTOR(S): Bennett, Kelly L.; Wolff, Edith A.; Aruffo, Alejandro A.; Greenfield, W. Brad

50613257

PATENT ASSIGNEE(S): Bristol-Myers Squibb Company, USA
SOURCE: PCT Int. Appl., 61 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9937317	A1	19990729	WO 1999-US1411	19990121 <--
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9934488	A	19990809	AU 1999-34488	19990121 <--
US 6559287	B1	20030506	US 1999-235230	19990121
PRIORITY APPLN. INFO.:			US 1998-72416P P	19980124
			WO 1999-US1411 W	19990121

AB Novel artificial proteoglycans containing a GAG assembly site and a control sequence required for assembly, method for enhancing the biol. activity of a glycosaminoglycan-binding protein using artificial proteoglycans, DNA constructs of artificial proteoglycans. The artificial proteoglycans of the present invention are useful for prepns. of adjuvants for vaccination, for targeting of chemokines to non-immunogenic tumor cells to enhance cellular anti-tumor response, for prepns. designed to help promote wound healing, and for treatment of immunol. disorders including rheumatoid arthritis, asthma, chronic obstructive pulmonary disorder, Lupus, inflammatory bowel disease, psoriasis, osteoarthritis, and HIV infection.

REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 16 OF 21 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1997:567826 CAPLUS
DOCUMENT NUMBER: 127:233174
TITLE: Anemia of chronic disorders in systemic autoimmune diseases
AUTHOR(S): Bertero, Maria Tiziana; Caligaris-Cappio, Federico
CORPORATE SOURCE: Dipartimento di Scienze Biomediche e Oncologia Umana, Cattedra di Immunologia Clinica, Universita di Torino, Turin, 10126, Italy
SOURCE: Haematologica (1997), 82(3), 375-381
CODEN: HAEMAX; ISSN: 0390-6078
PUBLISHER: Il Pensiero Scientifico
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review with 87 refs. that integrates the pertinent information from immunol. and hematol. to build a more complete understanding of anemia of chronic disorders (ACD) in systemic autoimmune diseases . ACD is a mild to moderate anemia characterized by decreased serum iron, decreased total iron-binding capacity and increased iron stores that occurs in a wide variety of diseases including cancer, chronic infections and inflammatory disorders. ACD is a parameter of disease activity in systemic autoimmune diseases. The severe inflammatory stimuli responsible

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for the pathophysiol. of these disorders lead to several systemic changes (referred to as chronic active phase response) through which the organism tries to cope with chronic tissue injuries. These reactions are brought about by inflammation-associated cytokines, like IL-6, IL-1, TNFα, TGFβ that regulate hepatic synthesis of acute phase proteins. Many cytokines involved in chronic acute phase response, including IL-1, TNFα, TGFβ, have an inhibitory activity on erythroid colony formation in vitro. In addition, circulating TNFα is elevated in rheumatoid arthritis (RA), IL-1β serum levels are significantly increased in RA with ACD and RA patients treated in vivo with antibodies (Abs) to TNFα show disease improvement, including an increase in Hb values. Reduced erythropoietin (EPO) activity, usually the result of reduced production, plays a role in the pathogenesis of ACD observed in systemic autoimmune diseases. Both the production and the action of EPO may fall under the control of IL-1 and IFN-γ. The most controversial and stimulating aspect of the pathogenesis of ACD in systemic autoimmune disorders is the role of iron metabolism and nitric oxide (NO), which contributes to the regulation of iron cellular metabolism Both iron deficiency and iron overload may influence the proliferation of B and T lymphocytes and differentially affect T helper (TH)-1 and TH-2 lymphocytes. Furthermore, TH-1 cytokines stimulate and TH-2-type cytokines inhibit NO production For these reasons, cell-mediated immunity may be expected to have influence on NO synthesis and on the mechanisms leading to iron accumulation in the reticuloendothelial system.

L7 ANSWER 17 OF 21 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1991:527011 CAPLUS
DOCUMENT NUMBER: 115:127011
TITLE: Method of treating inflammation with cartilage-inducing factor (CIF), and isolation and characterization of CIFs
INVENTOR(S): Bentz, Hanne; Ellingsworth, Larry; Armstrong, Rosa
PATENT ASSIGNEE(S): Collagen Corp., USA
SOURCE: U.S., 14 pp. Cont.-in-part of U.S. 4,806,523. CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 4971952	A	19901120	US 1988-263635	19881027 <--
US 4806523	A	19890221	US 1986-836672	19860306 <--
CA 1265445	A1	19900206	CA 1986-514887	19860729 <--
EP 213776	A2	19870311	EP 1986-306000	19860804 <--
EP 213776	A3	19890503		
EP 213776	B1	19921111		
R: AT, BE, CH, DE, FR, GB, IT, LI, LU, NL, SE				
EP 451439	A1	19911016	EP 1991-100255	19860804 <--
EP 451439	B1	19960313		
R: AT, BE, CH, DE, FR, GB, IT, LI, LU, NL, SE				
AT 82133	T	19921115	AT 1986-306000	19860804 <--
AT 135231	T	19960315	AT 1991-100255	19860804 <--
AU 8660875	A	19870212	AU 1986-60875	19860805 <--
AU 591513	B2	19891207		
US 5008240	A	19910416	US 1988-230330	19880809 <--
JP 06228005	A	19940816	JP 1993-337499	19931228 <--
JP 07098754	B	19951025		

US 5529982	A	19960625	US 1994-347592		19941130 <--
PRIORITY APPLN. INFO.:			US 1985-763337	B2	19850806
			US 1986-836672	A2	19860306
			EP 1986-306000	A	19860804
			US 1988-263635	A2	19881027
			US 1990-586363	B1	19900921
			US 1992-948063	B1	19920921

AB Inflammation, acute and/or chronic, is treated with a CIF, which may be administered locally or systemically, depending on the indication, and does not require coadministration of activator or cofactor for efficacy. CIF-A and -B were isolated from bovine bone, and partial amino acid compns. and amino-terminal sequences were determined [the chain sequence for a CIF homodimer (platelet-derived human transforming growth factor-β monomer) is also shown]. Both CIFs were potent inhibitors of human T-lymphocyte proliferation and also reduced production of IgM and IgG. In a rat arthritis model, treatment with CIF reduced inflammation significantly compared to the nontreated control group. The ability of CIF to suppress inflammation accompanying grafts or transplants were examined Also reported is histol. evaluation of rats having implants containing CIF-A/CIF-B and hematopoietic modulation by CIF-A.

L7 ANSWER 18 OF 21 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1991:527006 CAPLUS
DOCUMENT NUMBER: 115:127006
TITLE: Biologically active polypeptides based on transforming growth factor -β (TGF-β) sequences, and their use as immunosuppressive agents and antiinflammatory agents
INVENTOR(S): Burnier, John A.; Cianciolo, George J.
PATENT ASSIGNEE(S): Genentech, Inc., USA
SOURCE: PCT Int. Appl., 47 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9014359	A1	19901129	WO 1990-US1826	19900404 <--
W: CA, JP				
RW: AT, BE, CH, DE, DK, ES, FR, GB, IT, LU, NL, SE				
US 5061786	A	19911029	US 1989-356964	19890525 <--
CA 2057896	A1	19901126	CA 1990-2057896	19900404 <--
CA 2057896	C	20000606		
EP 473649	A1	19920311	EP 1990-908073	19900404 <--
EP 473649	B1	19950201		
R: AT, BE, CH, DE, DK, ES, FR, GB, IT, LI, LU, NL, SE				
ES 2070322	T3	19950601	ES 1990-908073	19900404 <--
US 5118791	A	19920602	US 1991-714462	19910613 <--
US 5268455	A	19931207	US 1992-824622	19920123 <--

PRIORITY APPLN. INFO.:	US 1989-356964	A	19890525
	WO 1990-US1826	W	19900404
	US 1991-714462	A3	19910613

OTHER SOURCE(S): MARPAT 115:127006
AB The title polypeptides exclude (a) a full-length mature TGF-β mol. or precursor TGF-β mol. or deletion variants of mature or precursor

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TGF-β mols. in which from about 1 to 10 amino acid residues have been deleted, (b) a polypeptide of the sequence: Cys-Val-Arg-Gln-Leu-Tyr-Ile-Asp-Phe-Arg-Lys-Asp-Leu-Gly-Trp-Lys, and (c) polypeptide of the sequence: Arg-Asn-Leu-Glu-Glu-Asn-Cys-Cys-Val-Arg-Pro-Leu-Tyr-Ile-Asp-Phe-Arg-Gln-Asp-Leu, said polypeptides comprising amino acid sequences that are based on conserved sequences in the family of TGF-β mols. Such polypeptides are particularly useful therapeutically as immunosuppressive agents when coupled to carrier proteins or crosslinked to form polymers. Thus, an albumin conjugate of a peptide of the invention inhibited proliferation of cultured mink lung cells, stimulated PGE2 production from fibroblasts, blocked binding of radiolabeled TGF-β to its receptor, inhibited proliferation of human lymphocytes in response to tetanus toxoid or other species, and inhibited human monocyte chemotactic response. The peptide conjugate was also effective in reduction of incidence of type II collagen-induced arthritis in mice.

L7 ANSWER 19 OF 21 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1991:447573 CAPLUS
DOCUMENT NUMBER: 115:47573
TITLE: Defective neutrophil function in the autoimmune mouse strain MRL/lpr. Potential role of transforming growth factor-β
AUTHOR(S): Gresham, Hattie D.; Ray, Carla J.; O'Sullivan, Frank X.
CORPORATE SOURCE: Res. Serv., Harry S. Truman VA Med Cent., Columbia, MO, 65201, USA
SOURCE: Journal of Immunology (1991), 146(11), 3911-21
CODEN: JOIMA3; ISSN: 0022-1767
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Patients with systemic autoimmune diseases such as SLE and rheumatoid arthritis have increased rates of morbidity and mortality caused by infection. Although this increased risk of infection has been primarily attributed to therapeutic immunosuppression, some reports exist of defective polymorphonuclear leukocytes (PMN) function in these patients. The purpose of the present work is to investigate the recruitment of PMN phagocytic function in a murine model of autoimmunity, the MRL/lpr mouse. PMN from MRL/lpr, but not from congenic MRL/n mice, exhibit a marked defect in the amplification of FcR-mediated phagocytosis stimulated by various inflammatory mediators. This defect is acquired and correlates with the onset of the autoimmune disease observed in this strain. In addition, MRL/lpr but not MLR/n PMN exhibit a defect in extravasation into the thioglycollate-inflamed peritoneum. Incubation of MRL/n PMN in MRL/lpr serum induces a defect in the amplification of PMN phagocytic function identical to that observed with MRL/lpr PMN. The activity in the serum that induces this defect is neutralized by an antibody to transforming growth factor (TGF)-β but not by control antibodies. Incubation of murine and human PMN with purified TGF-β induces an identical defect in stimulated FcR-mediated ingestion. TGF-β-treated MRL/n PMN fail to extravasate into the thioglycollate-inflamed peritoneum after injection into normal MRL/n recipient mice. Direct injection of TGF-β into MRL/n mice also reduces the percentage and number of PMN in the thioglycollate-stimulated peritoneal exudates of these mice. The defect in PMN extravasation and phagocytic function was not caused by failure of the defective PMN to modulate the expression of the adhesion mols., Mac-1 and Mel-14. Thus, defects in PMN function can be observed in a murine model of autoimmunity and spontaneous production of TGF-β possibly may play a crucial role in the pathogenesis of the defective PMN function in this animal model.

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L7 ANSWER 20 OF 21 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1990:605516 CAPLUS
DOCUMENT NUMBER: 113:205516
TITLE: Method of treating inflammatory disorders by reducing phagocyte activation with transforming growth factor (TGF)-β
INVENTOR(S): Nathan, Carl F.; Narachi, Michael A.
PATENT ASSIGNEE(S): Amgen, Inc., USA
SOURCE: PCT Int. Appl., 20 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9000900	A1	19900208	WO 1989-US3096	19890720 <--
W: AU, JP				
RW: AT, BE, CH, DE, FR, GB, IT, LU, NL, SE				
AU 8940560	A	19900219	AU 1989-40560	19890720 <--

PRIORITY APPLN. INFO.: US 1988-222579 A 19880720
WO 1989-US3096 A 19890720

AB Inflammatory disorders involving excessive phagocyte activation (e.g. adult respiratory distress syndrome, rheumatoid arthritis, asthma, emphysema, acute glomerular nephritis, inflammatory bowel disease) are treated by administration of TGF-β1 or TGF-β2. Thus, macrophages (source not stated) were activated in vitro with PMA. H2O2 release by the activated macrophages was 50% inhibited by incubation with 0.6 pM TGF-β1. This inhibition did not result from triggering of the respiratory burst, and was not accompanied by a major loss of phagocytic capacity.

L7 ANSWER 21 OF 21 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1989:51306 CAPLUS
DOCUMENT NUMBER: 110:51306
TITLE: Transforming growth factor -β in the treatment of inflammatory disorders
INVENTOR(S): Shepard, Harold Michael
PATENT ASSIGNEE(S): Genentech, Inc., USA
SOURCE: Eur. Pat. Appl., 9 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 269408	A2	19880601	EP 1987-310341	19871124 <--
EP 269408	A3	19890830		
R: AT, BE, CH, DE, ES, FR, GB, GR, IT, LI, LU, NL, SE				
JP 63211234	A	19880902	JP 1987-298859	19871126 <--

PRIORITY APPLN. INFO.: US 1986-935445 A 19861126
US 1987-116101 A 19871103

AB Methods and compns. are provided for the treatment of inflammatory disorders such as rheumatoid arthritis,

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inflammatory bowel disease, systemic lupus erythematosus and the like. Therapeutically EDs of Transforming Growth Factor-β are administered to patients in order to ameliorate these disorders. A patient with nondeforming rheumatoid arthritis with multiple joint involvement and a pos. latex fixation test for rheumatoid factor was treated with direct intraarticular injections of 50 ng human acid-activated TGF-β in isotonic saline in the left knee and 500 ng in 10 mL isotonic saline/day in the right knee, at 3 day intervals for 2 wk (totaling 5 injections). Less accessible joints were treated by an initial systemic (i.v.) loading infusion of 10 μg TGF-β in 5% dextrose, followed by monitoring of serum TGF-β concns. in order to confirm loading to about 1 ng TGF-β/mL serum. The patient's clin. condition improved as judged by diminution of redness and swelling, increased range of articular motion, increased hand grip strength, etc.

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FILE CONTAINS CURRENT INFORMATION.
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     FILE 'CAPLUS' ENTERED AT 15:27:37 ON 12 JAN 2007
L1         31500 S TRANSFORMING GROWTH FACTOR BETA
L2         14096 S L1 AND INHIBIT?
L3          4543 S L2 AND DISEASE?
L4          1028 S L3 AND INFLAMMATOR?
L5           148 S L4 AND ARTHRITIS?
L6            59 S L5 AND PY<2003
L7            21 S L6 AND DISORDER?

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FULL ESTIMATED COST                                       0.30      82.39

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
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